A Rare Case of Caecal Perforation with Ruptured Amoebic Liver Abscess.

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Received: April 2016 Accepted: April 2016

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ABSTRACT

Amoebic colitis is a common infection in all population groups across all geographical barriers and is an easily treatable disease in its routine manifestations. However, he has its set of complications that carry poor prognosis. Amoebic liver abscess is a common complication but its association with caecal perforation is rare, and carries worst prognosis. No survivors have been documented in the available literature and the result was pretty much the same in our study.

Keywords: Amoebic colitis, Liver abscess, Burst liver abscess, Colitis, E. Histolytica, Caecal perforation.

INTRODUCTION

Amoebiasis with its subset disease spectrum is a common parasitic infection distributed amongst all socioeconomic groups of patients and regions, producing diarrhoea, colitis, and amoebic liver abscess, predominantly in developing tropical countries. [1-3] Around 80% of the infected patients stay asymptomatic, while the other 20% develops disease. [1-3] Symptomatic patients of amoebiasis, result in 40,000 to 100,000 deaths worldwide each year. [3] Extra-amoebic colitis, likely amoebic liver abscess is the commonest cause contributing to mortality. The organism travels via portal vein to the liver and manifests as liver abscess.

This resultant abscess may rupture into the peritoneal, pleural and pericardial cavities. Intra-Peritoneal rupture of amoebic liver abscess is reported in around 6 – 9%.^[4] While bowel perforation from amoebiasis is very rare^[5,6] And it is extremely rare to have both these complications occurring simultaneously.

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CASE REPORT

A 58 year old chronic alcoholic and smoker male patient attended emergency surgery ward with complaint of pain in abdomen with associated intermittent fever, nausea & multiple episodes of

vomiting and anorexia for 15 days and obstipation for 4 days. Patient did not have history of loose motions, GI bleed or any medical co-morbidity.

On examination patient had a very poor general condition. He was conscious but drowsy, had pulse rate of 120/min, respiratory rate of 32/min and BP of 90/60 mm of Hg. Patient was icteric but had no pallor, cyanosis, oedema or lymphadenopathy. Chest examination showed generalised coarse crepitations.

Per abdomen, examination showed generalised peritonitis but the bowel sounds were present.

Patient was admitted in emergency ward and immediate IV fluid resuscitation performed with crystalloids and routine blood investigations were sent. These are as depicted in [Table 1].

Table 1: Investigations.		
S.N.	Investigation	Data
1	Hb	13.7 gm
2	TLC	35700 mm3,
		N82 L10 M04 E04
3	Blood urea	145 mg/dl
4	Serum creatinine	1.9 mg/dl
5	S. Amylase	31 IU/L
6	S. Lipase	12 IU/L
7	Na+	131
8	K+	4.4

After the patient was resuscitated and first shot of empirical intravenous antibiotics given, he was shifted for ultrasound abdomen and chest Xray. The chest Xray revealed bilateral costophrenic angle blunting and diffuse COPD changes. No free gas under diaphragms was seen.

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The Ultra sound abdomen suggested multiple liquefied liver abscess in right lobe of liver, largest being 200 cc, septated ascites and bilateral pleural effusion.

Patient was provisionally diagnosed as burst liver abscess with generalized peritonitis and planned for emergency laparotomy.

After proper preoperative preparation patient was shifted to OT. With induction of anaesthesia midline laparotomy performed and following intraoperative finding noted:

- Approx. 1000 ml bilious ascites with pus mixed anchovy coloured collection in perihepatic region.
- 2. 10X10 cm ruptured liver abscess in segment VII and VIII of liver [Figure 1].
- 3. 1X1 cm caecal perforation over anterior (antimesenteric) surface [Figure 2 & 3].



Figure 1: Burst liver abscess

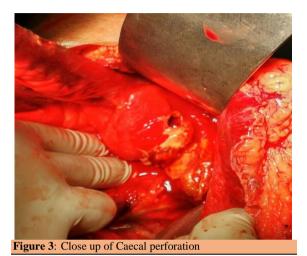


Figure 2: Caecal perforation

Thorough saline lavage of abscess and peritoneal cavity done. Right hemicolectomy followed by end ileostomy and mucous fistula performed. Drains

placed in abscess cavity, Morrison's pouch and the pelvis.

Postoperatively patient maintained vitals, but because of poor general condition, kept in ICU. Amoebic serology was sent in the immediate postoperative period and the results were positive. The patient did not improve and because of severe sepsis succumbed after 48 hours of surgery.



DISCUSSION

Entamoeba Histolytica is primarily an intra-luminal living organism of the large bowel. After consumption of an amoebic cysts via feco-oral route, cysts travel through the small intestine to the large intestine where they spring up the trophozoite stage that invades the bowel wall. Primarily the lesions are located in the large intestine though some may be seen in the terminal ileum. Initial lesions are more commonly localized in those fields where the colonic flow is slow, likely the cecum and recto sigmoid. The initial lesion is pinhead sized, but with rise in mucosal edema central ulceration results. Ulceration is mainly localized to mucosal epithelium and lamina propria. But when the ulcers progress to the muscularis propria they extend laterally along the axis of the intestine undermining the overlying mucosa. The communication of these laterally spreading ulcers with the intestinal lumen through a narrow mucosal defect creates the so called "flask like" ulcers. Adjacent ulcers may coalesce, leading to larger mucosal defects. Sometimes In advance cases, ulcers progresses beyond the muscularis propria and penetration results in a perforation of the intestinal wall. These perforations commonly occur in the cecum.^[7]

Amoebic liver abscess is the commonest extraintestinal complications of amebiasis. This form takes months to years to develop after the intestinal stage of the infection. The onset of hepatic symptoms may be gradual or rapid. Liver abscess is

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characterized by pain in the right upper quadrant, fever, anorexia, nausea, hepatomegaly and Liver tenderness etc.

Liver abscesses are most commonly located in the right lobe and may be single or multiple.

An early abscess initially presented with a minor area of parenchymal necrosis with greyish-brown cut surface. As far as area of necrosis increases in size, the center liquifies and a true cavity forms. These contents, most of the times sterile and non-pyogenic, become viscid and chocolate-coloured ("anchovy paste"). The incidence of intraperitoneal rupture of amoebic liver abscess is between 6 - 9 percent.

The both of above complications occurring simultaneously is very rare. Mukherjee *et al* documented single case of amoebic bowel perforation who died on the second post-operative day.^[8] Eggleston *et al* studied 26 cases of bowel perforations of the bowel, out of them 6 cases had un-ruptured liver abscess.^[9,10] All of them died shortly after surgery.

The surgery performed by us is in an elderly chronic alcoholic who had presented with very poor general condition with sepsis. And he succumbed to his illness 48 hours of surgery.

From treatment point of view drug of choice for amoebic colitis is metronidazole (given for 5 to 10 days). Treatment is divided into two groups of antiparasitic drugs: luminal and tissue agents. Asymptomatic phase of E. histolytica can be treated with luminal agents alone. Drugs used for the treatment of luminal infections are iodoquinol, diloxanide furoate, and paromomycin (5 to 20 days to eradicate colonization). The addition of broadspectrum antibiotics to the handling of acute amebic colitis may be appropriate if perforation is suspected. The possibility of coexisting bacteria causing dysentery must always be considered.

CONCLUSION

Simultaneous presentation of burst amoebic liver abscess with bowel perforation is an extremely rare entity and carries a very poor prognosis with almost 100% mortality.

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How to cite this article: Prajapati DK, Rampal K, Prajapati JM. A Rare Case of Caecal Perforation with Ruptured Amoebic Liver Abscess. Ann. Int. Med. Den. Res. 2016;2(4):6-8.

Source of Support: Nil, Conflict of Interest: None declared